

L1 106932 S "MYELIN DISORDER" OR "MULTIPLE SCLEROSIS" OR HTLV
 L2 413 S UNC-33 OR UNC33 OR ULIP OR CRMP OR "COLLAPSING RESPONSE MEDIA
 L3 0 S L1 AND L2
 L4 7 S L2 AND MYELIN
 L5 3 DUP REM L4 (4 DUPLICATES REMOVED)
 L6 34 S ULIP (S) CRMP
 L7 30 S L6 NOT PY>=2002
 L8 12 DUP REM L7 (18 DUPLICATES REMOVED)
 L9 6 S ULIP6
 L10 2 DUP REM L9 (4 DUPLICATES REMOVED)
 L11 32 S CRMP2
 L12 15 DUP REM L11 (17 DUPLICATES REMOVED)
 L13 7 S L12 NOT PY>=2002
 L14 7 S ULIP2
 L15 3 DUP REM L14 (4 DUPLICATES REMOVED)
 L16 3 S L15 NOT PY>=2002
 L17 0 S "METHOD OF TREATMENT" AND L2
 L18 0 S (MODULATING (P) GENE EXPRESSION) AND L2
 L19 6 S ULIP2 (S) CRMP2
 L20 0 S ULIP6/CRMP5
 L21 3 S ULIP6 (S) CRMP5
 L22 2 DUP REM L19 (4 DUPLICATES REMOVED)
 L23 1 DUP REM L21 (2 DUPLICATES REMOVED)
 L24 0 S L1 AND L11
 L25 36610 S DEMYELINATION OR MYELINATION
 L26 1 S L25 AND L2
 L27 0 S L25 AND L11
 L28 0 S L25 AND L6
 L29 7084 S L25 AND L1
 L30 0 S L29 AND CRMP

=>

ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
 ACCESSION NUMBER: 2001500585 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11549731
 TITLE: Isolation and expression pattern of human Unc-33-like phosphoprotein 6/collapsin response mediator protein 5 (Ulip6/CRMP5): coexistence with **Ulip2/CRMP2** in **Sema3a**- sensitive oligodendrocytes.
 AUTHOR: Ricard D; Rogemond V; Charrier E; Aguera M; Bagnard D; Belin M F; Thomasset N; Honnorat J
 CORPORATE SOURCE: Institut National de la Sante et de la Recherche Medicale U 433, Institut Federatif des Neurosciences de Lyon, Hopital Neurologique, 69003 Lyon, France.
 SOURCE: Journal of neuroscience : official journal of the Society for Neuroscience, (2001 Sep 15) 21 (18) 7203-14.
 Journal code: 8102140. ISSN: 1529-2401.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 OTHER SOURCE: GENBANK-AF264015
 ENTRY MONTH: 200110
 ENTRY DATE: Entered STN: 20010911
 Last Updated on STN: 20011008
 Entered Medline: 20011004

AB The Unc-33-like phosphoprotein/collapsin response mediator protein (Ulip/CRMP) family consists of four homologous phosphoproteins considered crucial for brain development. Autoantibodies produced against member(s) of this family by patients with paraneoplastic neurological diseases have made it possible to clone a fifth human Ulip/CRMP and characterize its cellular and anatomical distribution in developing brain. This protein, referred to as Ulip6/CRMP5, is highly expressed during rat brain development in postmitotic neural precursors and in the fasciculi of fibers, suggesting its involvement in neuronal migration/differentiation and axonal growth. In the adult, Ulip6/CRMP5 is still expressed in some neurons, namely in areas that retain neurogenesis and in oligodendrocytes in the midbrain, hindbrain, and spinal cord. **Ulip2/CRMP2** and Ulip6/CRMP5 are coexpressed in postmitotic neural precursors at certain times during development and in oligodendrocytes in the adult. Because **Ulip2/CRMP2** has been reported to mediate semaphorin-3A (**Sema3A**) signal in developing neurons, in studies to understand the function of Ulip6/CRMP5 and **Ulip2/CRMP2** in the adult, purified adult rat brain oligodendrocytes were cultured in a **Sema3A**-conditioned medium. Oligodendrocytes were found to have **Sema3A** binding sites and to express neuropilin-1, the major **Sema3A** receptor component. In the presence of **Sema3A**, these oligodendrocytes displayed a dramatic reduction in process extension, which was reversed by removal of **Sema3A** and prevented by anti-neuropilin-1, anti-Ulip6/CRMP5, anti-**Ulip2/CRMP2** antibodies, or VEGF-165, another neuropilin-1 ligand. These results indicate the existence in the adult brain of a **Sema3A** signaling pathway that modulates oligodendrocyte process extension mediated by neuropilin-1, Ulip6/CRMP5, and **Ulip2/CRMP2**, and they open new fields of investigation of neuron/oligodendrocyte interactions in the normal and pathological brain.

L22 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
 ACCESSION NUMBER: 2001142580 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11085871
 TITLE: Differential expression of collapsin response mediator proteins (CRMP/ULIP) in subsets of oligodendrocytes in the postnatal rodent brain.
 AUTHOR: Ricard D; Stankoff B; Bagnard D; Aguera M; Rogemond V; Antoine J C; Spassky N; Zalc B; Lubetzki C; Belin M F; Honnorat J

CORPORATE SOURCE: INSERM U433 Hopital Neurologique, Lyon, France.
 SOURCE: Molecular and cellular neurosciences, (2000 Oct) 16 (4)
 324-37.
 Journal code: 9100095. ISSN: 1044-7431.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200103
 ENTRY DATE: Entered STN: 20010404
 Last Updated on STN: 20010404
 Entered Medline: 20010308

AB The family of collapsin response mediator protein/Unc-33-like protein (CRMP/Ulip), composed of four homologous members, is specifically and highly expressed in the nervous system during embryonic neuronal development and dramatically down-regulated in the adult. Members of this family have been proposed to be part of the semaphorins signal transduction pathway involved in axonal outgrowth. Here, we show by in situ hybridization and immunohistochemistry that **CRMP2/Ulip2**, and to a lesser extent CRMP3/Ulip4, are expressed in immature and mature oligodendrocytes, but not in astrocytes. Transcripts encoding the other CRMP/Ulip members are also detectable by RT-PCR in highly purified mature oligodendrocytes. Interestingly, in the adult, the protein **CRMP2/Ulip2** is mainly detectable in subsets of oligodendrocytes distributed according to an increasing rostrocaudal gradient, with the largest number of positive cells being present in the brain stem and spinal cord. In cultures of highly purified oligodendrocytes, however, **CRMP2/Ulip2** was detectable in all the cells. Addition of Semaphorin 3A in the culture medium completely inhibited the emergence of oligodendrocyte processes suggesting that, as in neurons, a Semaphorin 3A signaling pathway mediated via **CRMP2/Ulip2** may be involved in the regulation of oligodendroglial process outgrowth.

=> d his

(FILE 'HOME' ENTERED AT 20:13:08 ON 14 OCT 2004)

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 20:13:18 ON 14 OCT 2004

L1 106932 S "MYELIN DISORDER" OR "MULTIPLE SCLEROSIS" OR HTLV
 L2 413 S UNC-33 OR UNC33 OR ULIP OR CRMP OR "COLLAPSING RESPONSE MEDIA
 L3 0 S L1 AND L2
 L4 7 S L2 AND MYELIN
 L5 3 DUP REM L4 (4 DUPLICATES REMOVED)
 L6 34 S ULIP (S) CRMP
 L7 30 S L6 NOT PY>=2002
 L8 12 DUP REM L7 (18 DUPLICATES REMOVED)
 L9 6 S ULIP6
 L10 2 DUP REM L9 (4 DUPLICATES REMOVED)
 L11 32 S CRMP2
 L12 15 DUP REM L11 (17 DUPLICATES REMOVED)
 L13 7 S L12 NOT PY>=2002
 L14 7 S ULIP2
 L15 3 DUP REM L14 (4 DUPLICATES REMOVED)
 L16 3 S L15 NOT PY>=2002
 L17 0 S "METHOD OF TREATMENT" AND L2
 L18 0 S (MODULATING (P) GENE EXPRESSION) AND L2
 L19 6 S ULIP2 (S) CRMP2
 L20 0 S ULIP6/CRMP5
 L21 3 S ULIP6 (S) CRMP5
 L22 2 DUP REM L19 (4 DUPLICATES REMOVED)
 L23 1 DUP REM L21 (2 DUPLICATES REMOVED)

=> d 123 ibib abs total

L23 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001500585 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11549731
TITLE: Isolation and expression pattern of human Unc-33-like phosphoprotein 6/collapsin response mediator protein 5 (**Ulip6/CRMP5**): coexistence with Ulip2/CRMP2 in Sema3a- sensitive oligodendrocytes.
AUTHOR: Ricard D; Rogemond V; Charrier E; Aguera M; Bagnard D; Belin M F; Thomasset N; Honnorat J
CORPORATE SOURCE: Institut National de la Sante et de la Recherche Medicale U 433, Institut Federatif des Neurosciences de Lyon, Hopital Neurologique, 69003 Lyon, France.
SOURCE: Journal of neuroscience : official journal of the Society for Neuroscience, (2001 Sep 15) 21 (18) 7203-14. Journal code: 8102140. ISSN: 1529-2401.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF264015
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20010911
Last Updated on STN: 20011008
Entered Medline: 20011004

AB The Unc-33-like phosphoprotein/collapsin response mediator protein (Ulip/CRMP) family consists of four homologous phosphoproteins considered crucial for brain development. Autoantibodies produced against member(s) of this family by patients with paraneoplastic neurological diseases have made it possible to clone a fifth human Ulip/CRMP and characterize its cellular and anatomical distribution in developing brain. This protein, referred to as **Ulip6/CRMP5**, is highly expressed during rat brain development in postmitotic neural precursors and in the fasciculi of fibers, suggesting its involvement in neuronal migration/differentiation and axonal growth. In the adult, **Ulip6/CRMP5** is still expressed in some neurons, namely in areas that retain neurogenesis and in oligodendrocytes in the midbrain, hindbrain, and spinal cord. Ulip2/CRMP2 and **Ulip6/CRMP5** are coexpressed in postmitotic neural precursors at certain times during development and in oligodendrocytes in the adult. Because Ulip2/CRMP2 has been reported to mediate semaphorin-3A (Sema3A) signal in developing neurons, in studies to understand the function of **Ulip6/CRMP5** and Ulip2/CRMP2 in the adult, purified adult rat brain oligodendrocytes were cultured in a Sema3A-conditioned medium. Oligodendrocytes were found to have Sema3A binding sites and to express neuropilin-1, the major Sema3A receptor component. In the presence of Sema3A, these oligodendrocytes displayed a dramatic reduction in process extension, which was reversed by removal of Sema3A and prevented by anti-neuropilin-1, anti-**Ulip6/CRMP5**, anti-Ulip2/CRMP2 antibodies, or VEGF-165, another neuropilin-1 ligand. These results indicate the existence in the adult brain of a Sema3A signaling pathway that modulates oligodendrocyte process extension mediated by neuropilin-1, **Ulip6/CRMP5**, and Ulip2/CRMP2, and they open new fields of investigation of neuron/oligodendrocyte interactions in the normal and pathological brain.

=>